The Journal of HEREDITY

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All About Fingerprints

Sex Mosaics in Wasps

Deafness Inherited

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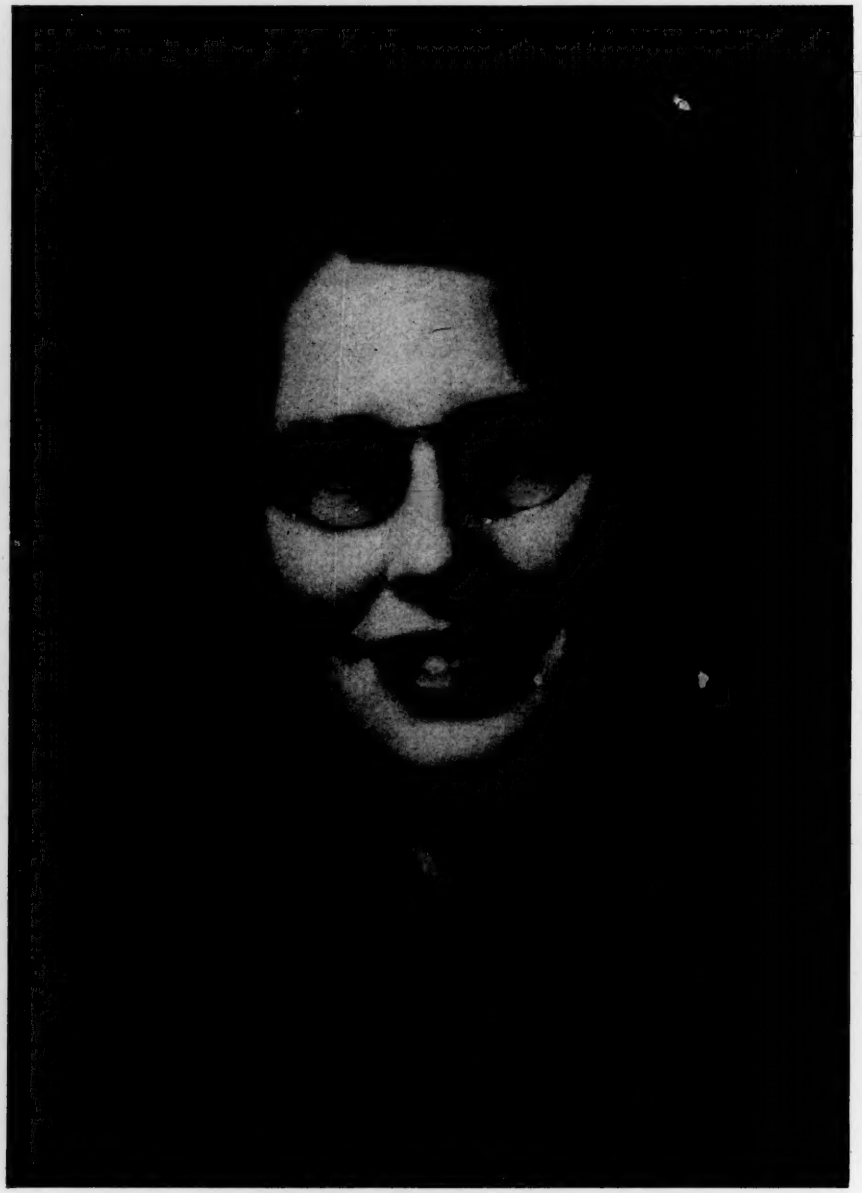


BARBARA STODDARD BURKS
1902-1943

Her untimely death ended a brilliant career in psychology and genetics. In her memory as a brilliant research worker and delightful companion, the friends of Barbara Burks have established a memorial fund, as announced on page 376.

ANDROGENESIS IN THE PARASITIC WASP HABROBRACON

P. W. WHITING
University of Pennsylvania, Philadelphia, Pa.

IN 1935 there was published in this JOURNAL[11] a discussion of sex determination in bees and wasps including a review of data and theories as applied to the parasitic wasp *Habrobracon juglandis* (Ashmead). An allelic pair of sex factors, *X* and *Y*, was postulated with the female heterozygous, *XY*, the haploid males of two types, *X* and *Y*, and corresponding diploid males, *XX* and *YY*. Sex-linkage of the mutant gene *fused* was shown for crosses of "related" parents in which diploid males occur. Biparental offspring from "unrelated" parents are females only, the homozygous combinations being absent.

To explain this lack of diploid males a hypothesis of differential maturation was suggested. Further data have shown that this hypothesis is incorrect and that the true explanation lies in the fact that there is a series of multiple sex alleles,—*xa, xb, xc, xd*, etc., rather than a single pair, *X* and *Y*.[12]

Any heterozygous combination, *xa/xb, xa/xc, xc/xd*, etc., is female; any haploid or homozygote is male. There have now been shown nine alleles in the series and probably many more will be found after tests of wild stocks have been made.

To illustrate what is known about sex-linkage of fused and "relationship" of parents, let us consider what happens when a white-eyed female heterozygous for fused and having sex alleles *xg/xh* is crossed with fused males with various sex alleles and with black wild type eye color. There are two types of such females according as fused is in the *xg* or the *xh* chromosome. Let us assume the latter, $\frac{wh\ xg\ +}{wh\ xh\ fu}$, and that females of this type are crossed with "related" *xg fu* and *xh fu* males and with "unrelated" *xa fu* males (Table I).

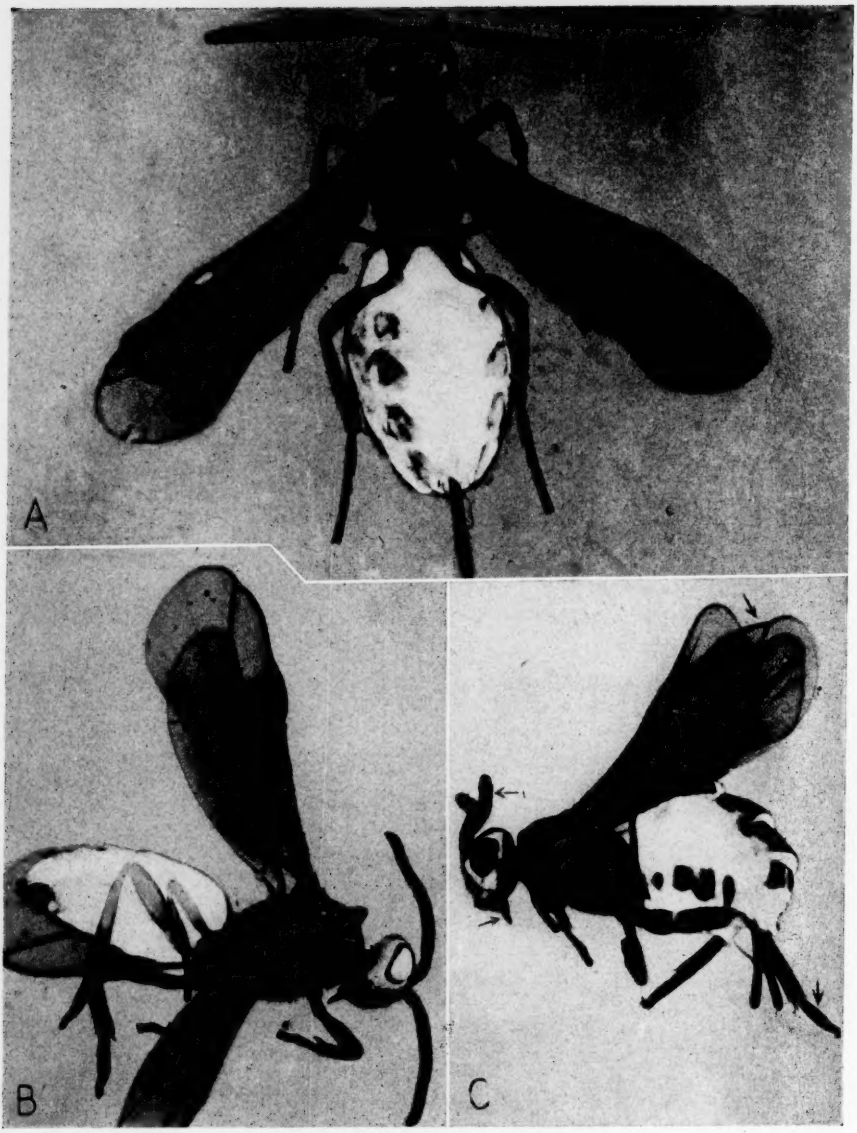
If the mother is unmated all her offspring will be white-eyed males, fused and non-fused in about equal numbers. Approximately one-third of the eggs of a mated female remain unfertilized giving a similar result. Among the diploid biparental offspring, the males are of relatively low viability so that there are many unhatchable eggs and fecundity is reduced. Such males occur only in the two-allele fraternities or, as previously expressed, when the parents are "related," the father having one of the sex alleles present in the mother. If the parents are "unrelated" the fraternity is three-allele, the father having a sex allele different from either of those in the mother. All diploid offspring will then be female because heterozygous and sex-linkage of fused is masked since we cannot separate the *xg/xa* females from the *xh/xa* by inspection.

Stocks having different sex alleles have been marked with different non-allelic recessive eye colors. Crosses between the stocks then produce only wild type black-eyed females from fertilized eggs. The wild type allele to the recessive eye color and also the gene fused may be introduced into any stock by crossing and recombination with the sex alleles of that stock. Diploid males appear as evidence of this recombination and the sex alleles of each stock may be shown to be linked with fused. In crosses between the stocks no diploid males appear and sex-linkage is masked as expected, fused and wild type females appearing in equal numbers.

Modifiers of Fused

The gene fused affects many traits (Figures 1*B-C*, and 2). Segments of antennae and of tarsi are much reduced and fused together. There is reduction in

The writer is indebted to the Rockefeller Foundation for a grant aiding in the investigations reported in this paper.



NORMAL AND "FUSED" WASPS

Figure 1

A—Wild type female heterozygous for white eyes and for the sex-linked gene fused. Her unfertilized eggs will develop into four types of males: wild type, white-eyed, fused and white-eyed fused (See *B*). If she is mated with a fused male having one of the same sex factors that she possesses, she will also produce wild type and fused females and wild type and fused diploid males in ratios showing sex-linkage as explained in the text. *B*—White-eyed fused haploid male. The antennae are longer than in the fused female (See *C*). Sex difference is shown in the lighter integument of the abdomen as well as in the sting. The finer texture of the wing surface is due to the more numerous microchaetae or minute bristles, each one of which corresponds to a single cell. The cells are smaller and more numerous in the haploid male than in the diploid female. *C*—Fused female. Notice the short antennae, tarsi and palpi with segments fused together, also the indentation near the tip of the costal margin of the primary wing. These traits are all characteristic of fused and segregate together as a single hereditary unit.

number of segments of maxillary and labial palpi. A notch occurs near the tip of the primary wing. Malformation of legs involves female sterility since the females are unable to grasp, sting and feed on the host caterpillars and thus obtain nutriment necessary for development of eggs.

The traits are very variable due to modifying factors in different stocks. The stock with sex alleles *xc/xd*, for example, has plus modifiers increasing the fused effects to such an extent that it is very difficult to maintain the gene in this combination. Legs and antennae become very short and bent, wings are very frequently wrinkled and many wasps are unable to eclose (escape) from their cocoons. In contrast the *xa/xb* stock has minus modifiers counteracting the fused effects so that fused wasps graded to *xa/xb* have relatively long straight legs and antennae and are able to eclose with little difficulty. We may speak of eugenic fused and of dysgenic fused according as minus or plus modifiers for the traits are present.

That these modifiers are not the sex alleles themselves was shown in the process of combining fused with sex alleles *xg/xh* for example. A "eugenic" fused male (*xa* or *xb*) was crossed with an *xg/xh* female. F_2 fused males were of a variety of types ranging from eugenic to very dysgenic. Matings were then made between these F_2 fused males and *xg/xh* females. A fraternity was obtained (one among 12) which included diploid males, indicating that the gene fused had recombined by crossing-over, so that it was now united with one of the alleles *xg* or *xh*. Further grading to *xg/xh* stock produced fused so dysgenic that it was very difficult to maintain. A cross was then made back to the *xa/xb* stock and well-formed F_2 fused males were obtained most of which still had the *xg/xh* sex alleles as shown by subsequent test.

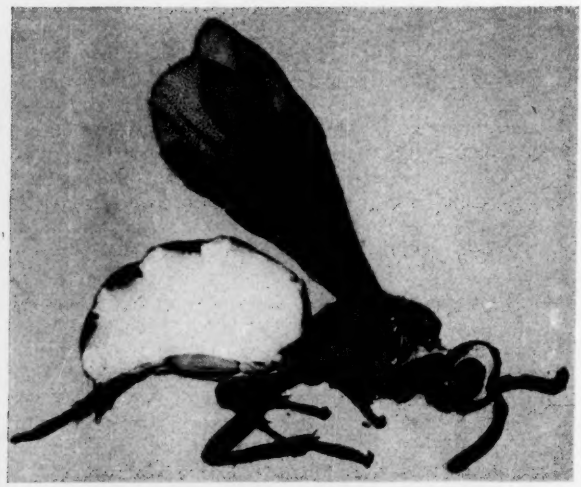
Instead of fused being graded to the *xg/xh* stock with plus modifiers, the method was then followed of inbreeding and selection. Daughters of eugenic fused males were crossed with eugenic fused males, either their brothers or closely related males in the *xg/xh* material. There resulted very shortly a stock in which practically all fused wasps were well formed, highly viable and from which there was no difficulty in obtaining matings. The same method gave similarly good results in combination with the alleles *xe/xf* and with *xa/xi* in a stock having one allele in common with the *xa/xh* stock.

Factors modifying fused do not affect all traits similarly to produce a general eugenic or a general dysgenic effect. For example, a group of related fraternities was obtained in which fused males had long well formed antennae and wings practically free from the notch but with legs so malformed that they were unable to walk about or to mate (Figure 1*B*). Fused wasps in the *xa/xb* stock have rather thin and long appendages, while those of *xe/xf* are short but nevertheless very well formed. Fused wasps in the *xg/xh* stock and also in *xa/xi* have long relatively thick appendages. Fused segregating in a yellow, *Y*, stock had very long antennae showing considerable segmentation and in other lines the tarsi have displayed a similar tendency. No instances have yet been obtained, however, approximating wild type or at all difficult to classify.

Haploid fused males have antennae much longer and somewhat thinner (Figure 1*B*) than fused females (Figure 1*C*). Antennae of diploid fused males (Figure 2) average but little longer than those of the females and show much overlapping. These differences may be seen readily in inbred stocks where variation due to modifying factors is reduced to a minimum. In mixed material the haploid males may overlap not only with the diploid males but even with the females. Reduced antennal length of diploid males should not be regarded as intersexuality but as due to the doubled chromosome number.

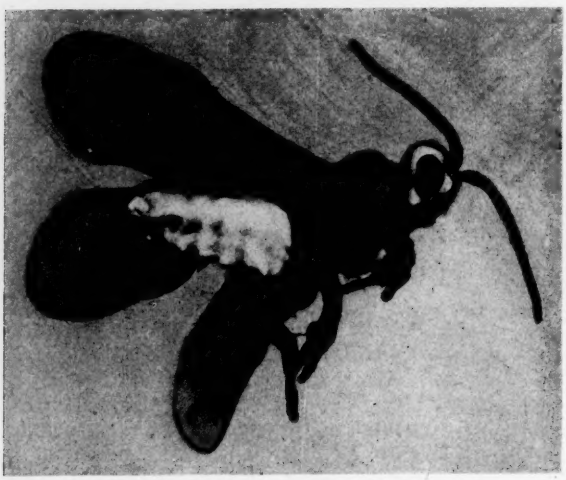
Irregular Types

As stated above, unmated females regularly produce haploid males only. Mated females produce, in addition to



FUSED DIPLOID MALE
Figure 2

The antennae are almost as short as in the fused female. This is not an intersexual character but is due to the doubled chromosome number. Notice the very coarse texture of the wing surface due to wide scattering of the microchaetae. Cells of the diploid male are much larger than those of the diploid female and very much larger than those of the haploid male.



"DYSGENIC" LEGS AND "EUGENIC" WINGS
Figure 3

Fused is subject to many modifying factors differing in different stocks. Plus modifiers increase the fused effects causing low viability and misshapen "dysgenic" specimens. Minus modifiers tend to counteract the fused effects, having a "eugenic" influence. Different structures are affected by different modifiers. This specimen is from a group of fraternities with legs very "dysgenic" but antennae and wings very "eugenic." The wings are well expanded and the characteristic notch is absent.

these, females in three-allele fraternities, females and diploid males in two-allele fraternities with diploid males less frequent than females because of low viability.

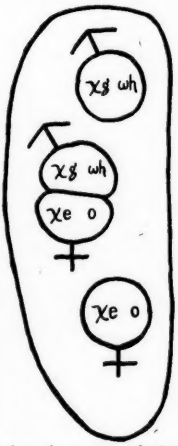
Irregular types, constituting small percentages of the totals, may be considered according as they are (1) impaternate (from unfertilized eggs) or as the matings involve (2) three sex alleles or (3) two sex alleles (Table II).

(1) The word impaternate was coined by Ray Lankester[1] who states: "Clearly the word 'parthenogenetic' has been, and must be, used to describe the virgin mother, and therefore cannot at the same time be applied to her offspring without causing confusion. It seems to me that the word 'impaternate' or 'fatherless' should be used for the offspring. I have failed to excogitate any other term which will so well meet the case."

In *Habrobracon*, haploid mosaic males occur among the offspring of females heterozygous for various traits. They develop from unfertilized binucleate eggs in

HOW MOSAICS ARISE

Figure 4



Fertilization in an egg of *Habrobracon* such as might have developed into a "trinucleate" gynander, like freak No. 935 (Figures 5 and 6). Two haploid maternal nuclei are present, ♀ — ♀, each bearing the sex allele *xe* and the recessive gene for orange eye. *o*. Two sperm nuclei ♂ — ♂, have entered the egg carrying the sex allele *xg* and the paternal white eye color gene *wh*. The diploid nucleus resulting from syngamy $\frac{xg + wh}{xe\ o\ +}$ will produce female parts of the gynander because its two sex alleles are different. The haploid nuclei give rise to male parts.

In all previous work, with very rare exceptions, development from the sperm nucleus has not been found. This phenomenon, called *androgenesis*, appears in crosses involving a recently studied stock, *CHB-VI-o*. Many gynanders have previously been found resulting from diploid biparental and haploid maternal nuclei. The recessive white eyes of freak 935 indicate androgenesis of the head region.

Mothers described in this study were in all cases from the orange-eyed stock *CHB-VI-o*, having one sex allele, *xe*, the other as yet undetermined. Fathers were mottled-eyed, *mo*, with sex alleles *xc* or *xd*, white-eyed, *wh*, with *xg* or *xh*, or red-eyed, *rd*, with *xe* or *xf*.

which the two nuclei carry different genes. Their frequency has been variously estimated as one in 500 to one in 5,000. Frequency of binucleate eggs must be considerably higher than this, however, since many mosaics are missed through oversight or because differentiating traits are lacking. Under favorable conditions mosaic males may range as high as one per cent. This percentage is taken as a rough guess for the frequency of binucleate eggs (Table II).

In a few cases in which the mothers were heterozygous for more than one factor, mosaic males have occurred showing three different combinations of the characters involved. These mosaics must have come from *trinucleate* eggs.[10]

The origin of the two nuclei in binucleate eggs is not known. If they are two of the four produced by reduction of a single oöcyte, they should contain different sex alleles twice as frequently as similar alleles: 1 *xa* - *xa*: 1 *xb* - *xb*: 4 *xa* - *xb*. This ratio is assumed in Table II. Males with feminized genitalia (gynandroid) probably constitute about ten per cent of the *xa* - *xb* mosaics.[14]

Impaternate females have occasionally been found, incidence varying with stock (0 to 1%).[3] Cytological evidence indicates that they owe their origin to doubling of chromosome number in certain cysts of the oögonia.[4] During investigations involving 291 impaternate females, two impaternate gynanders occurred, presumably from binucleate eggs with one nucleus diploid.[3] Impaternate diploid males may be expected but have not been found.[4]

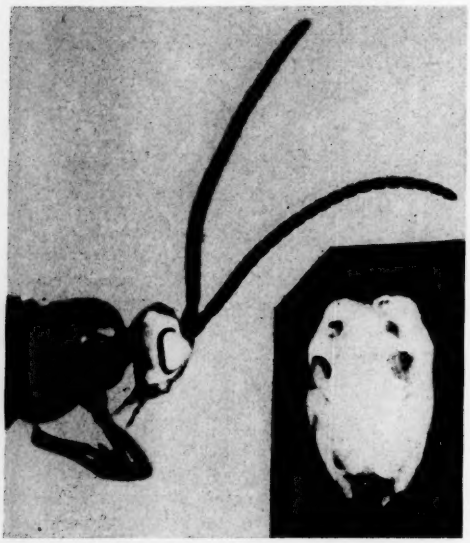
(2) Among fertilized eggs of *Habrobracon* about one per cent are fertilized by two sperm (dispermic). Table II shows what might be expected from one per cent binuclearity and one per cent dispermy if androgenesis (haploid development from sperm nucleus) could function as effectively as gynogenesis (haploid development from unfertilized egg nucleus) and if male diploidy did not decrease viability.

In three-allele fraternities there should be 0.99 per cent gynanders with male parts gynogenetic (*G*), 0.99 per cent with male parts androgenetic (*A*) and 0.01 per cent mosaic females.

(3) In two-allele fraternities as compared with three-allele, half the females are replaced by diploid males of low viability and correspondingly among the mosaics, half the female parts are replaced by diploid male parts. Haplo-diploid males, 0.495 per cent, mosaic diploid males, 0.0017 per cent, and gynanders with biparental male parts, 0.0067 per cent, should then appear in addition to the types occurring in three-allele fraternities.

Androgenesis of male parts should occur as frequently as gynogenesis among mosaics from fertilized eggs.

Several hundred mosaics of the two "orthodox" types, haploid mosaic males from unfertilized eggs and gynanders



ANDROGENIC HEAD
Figure 5

Profile of head and thorax and ventral view of abdomen of white-eyed sex mosaic or gynander, No. 935 (See Figure 6). The specimen is predominantly male with female regions restricted to certain parts of the abdomen. Deriving its gonads from its orange-eyed mother, it bred like an orange male. The head, with white eyes, was developed from an extra sperm nucleus, androgenesis. The long jointed antennae are characteristic of the normal male. This photo shows the long jointed palpi which are to be contrasted with the short palpi of fused wasps.

with gynogenetic male parts, have been found. Frequency of the latter among offspring from all fertilized eggs is roughly similar to that of the former among offspring from unfertilized eggs of heterozygous mothers.

Three mosaic females (nos. 279, 313, 469) and four mosaic diploid males (nos. 318, 337, 344, 411) have been reported[6] as probably due to spontaneous chromosome elimination in development. Three females were reported[2] as mosaic due to x-ray induced elimination.

Two haplo-diploid male mosaics with gynogenetic haploid parts and biparental diploid parts have been reported.[10] One (no. 544) bred as a haploid male. The other (no. 675) had mosaic gonads, producing many haploid and a few diploid sperm.

A single mosaic female[10] and two gynanders with male parts showing the dominant paternal traits (nos. 325, 438)[5] are probably best explained as developed from binucleate eggs with each nucleus fertilized by a different sperm (dispermy).

The low frequency of haplo-diploid males reported as compared with gynanders may be attributed to their low viability, to difficulty in recognizing them and to their occurrence in the two-allele fraternities only.

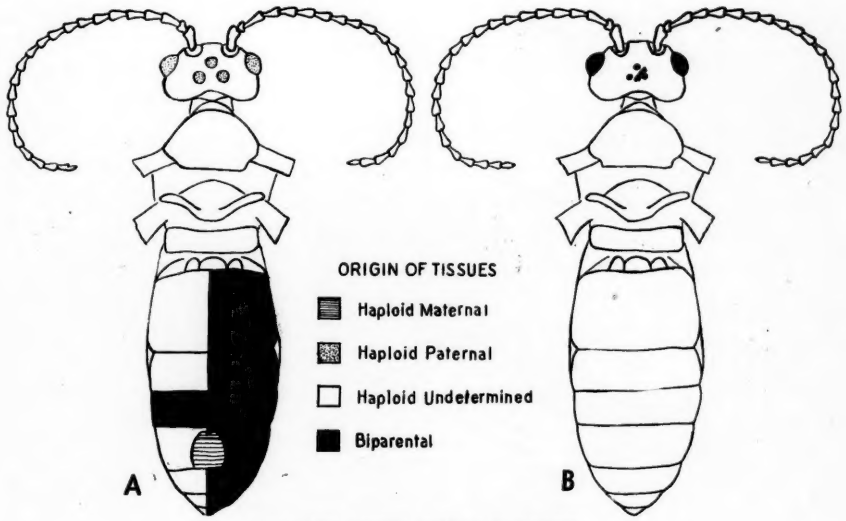
Among the mosaics above reported nos. 313, 325, 337, 438 and 469 might possibly be explained as having androgenetic haploid parts.

An unreported example of androgenesis, found by Arnold M. Clark, among the offspring of a black cantaloup honey female crossed with a lemon long veinless male, is a male showing the traits lemon, long and veinless. Of two gynanders reported,[10] one (no. 523) with female abdomen had male head bearing the recessive paternal trait orange eyes. The other (no. 640), from a gynoid female (*gy*) crossed with an eyeless (*el*) male, had the two different kinds of recessive male tissue, indicating that the head and a small part of the abdomen were from the sperm nucleus (*el*), while almost half of the abdomen was from an unfertilized egg nucleus (*gy*), the remainder biparental (female). Both egg binuclearity and dispermy are indicated.

The extreme rarity of reported cases of androgenesis stands in contrast to the great number of gynanders observed with gynogenetic male parts. Since dispermy is relatively frequent, functioning of the second male nucleus must be prevented by some condition that does not affect a second egg nucleus.

Stock with Androgenetic Tendency

The writer has recently received from Dr. C. H. Bostian a number of closely



MALES WITH FEMALE PARTS

Figure 6

A—No. 935, a "trinucleate" gynander showing biparental female regions in the abdomen. The body is in general haploid male, the gonads bearing orange from the mother, the eyes white from the father. *B*—No. 936, A "male" with black biparental female eyes. Small size of ocelli show them to be female and asymmetry of integumental pigment in ocellar region indicates a small patch of female tissue on the right. Despite the long male antennae, reactions of this wasp were entirely female indicating a female brain. There was no trait other than eye color,—maternal orange, paternal mottled, to indicate whether the body was androgenetic, gynogenetic or mixed.

inbred stocks which are being tested for their sex alleles. In addition to the 68 wild type females and 32 orange males expected from an orange-eyed mother (Bostian stock *CHB-IX-o*) crossed with a mottled-eyed father with sex alleles *xc* or *xd* (stock *xc/xd-mo*), there was an androgenetic mottled male (no. 934). In addition to 1023 wild type females and 608 orange males from orange females of another orange Bostian stock (*CHB-VI-o*) crossed with stock *xc/xd-mo* males were four mottled gynanders (nos. 934.2, 937, 939, 940), one gynander mosaic for mottled (no. 949) and three wild type wasps at first thought to be male (nos. 936, 941, 942). Wild type males are unexpected because several other tests indicate crosses of Bostian stocks to *xc/xd-mo* to be three-allele, from which all biparentals should be female.

CHB-VI-o females were also crossed with stock *xg/xh*-white males, yielding one white gynander (no. 935), one orange gynander (no. 938), one gynander mosaic for white (no. 950) in addition to 805 wild type females and 459 orange males. Crosses of *CHB-VI-o* females with stock *xg/xh* males are also three-allele lacking diploid males.

CHB-VI-o females were crossed with stock *xe/xf*-red males. Since these stocks have one sex allele in common, *xe*, *CHB-VI-o* may be designated *xe/x?-o*. Eight fraternities included, in addition to 328 wild type females and 257 orange males, one orange gynander (no. 934.3). Ten included, in addition to 260 wild type females, 269 orange males and 45 wild type males, one red male (no. 934.4), one male mosaic for red and fused (no. 943) (the father in this case was red fused), and one male mosaic for red (no. 951). The 45 wild type males in the last group are diploid biparentals,

ANDROGENETIC MALE AREAS
Figure 7

Gynander No. 950. The white mark in the right eye, the colorless large ocelli and most of the right antenna are male, derived from a second sperm nucleus, androgenesis. The shorter left antenna, the black eyes and the large abdomen with heavy integument and sting are female.

xe/xe. Ratio of females to haploid males is higher as expected in the fraternities yielding no diploid males since some at least are three-allele.

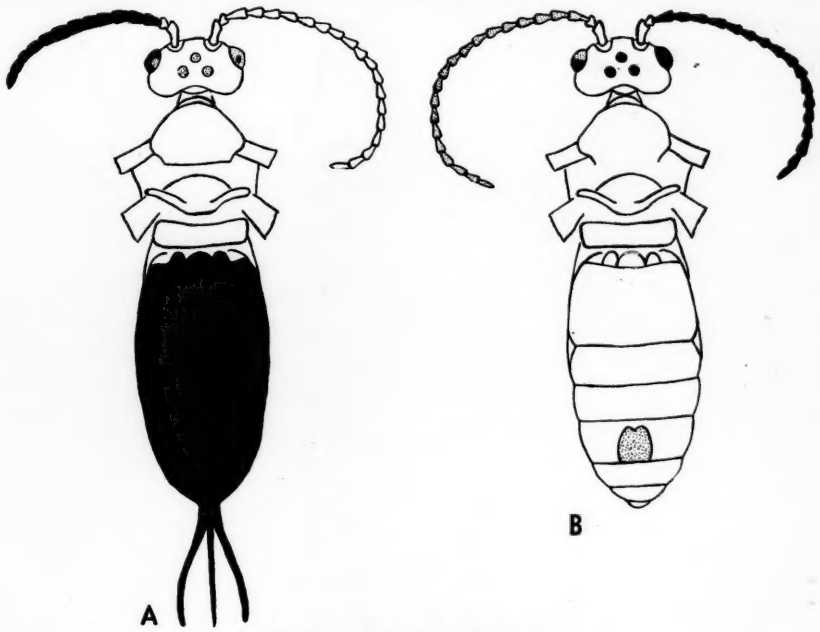
Offspring from fertilized eggs laid by *CHB-VI-o* mothers total 2476 (expected ♀ + 2416, ♂ + 45, irregular 15) among which the irregular are 0.606%. The mottled male (no. 934) from *CHB-IX-o* mother indicates that this stock, related to *CHB-VI-o*, also carries the androgenetic tendency.

The frequency of these irregular types, most or perhaps all of which contain androgenetic tissue, stands in contrast to the extreme rarity of androgenesis among mosaics of Habrobracon in general. A genetic difference is clearly indicated.

Detailed study was made of twelve of the irregular specimens with orange-eyed mothers of stock *CHB-VI-o*. Sex alleles of the paternal stocks, *xc/xd* and *xg/xh*, differ from those of maternal *xe/x?*, so that no diploid male tissue is expected. Only when the father has *xe* (from the *xe/xf* stock) can haplo-diploid males be produced. The paternal eye color white (*wh*), mottled (*mo*) or red (*rd*) or the maternal orange (*o*) may appear either in the eyes of the specimen or in its breeding tests. Orange and red are non-autonomous and may be darkened by proximity to non-orange or non-red tissue.[13] Diploid eyes will in all cases be wild type black since the mutant genes are non-allelic recessives. White and mottled ocelli lack pigment; orange and red normally have a small amount of reddish pigment which may be darkened considerably by proximity to the wild type allele. Wild type ocelli are dark brown.

Asymmetry of wild type body color indicates mosaicism[7] since coloration in non-mosaic wasps is very symmetrical and there are multiple factor differences determining amount and distribution of color.

No. 935, with stock *xg/xh-wh* father, was at first taken for a white-eyed male having head, thorax, wings and legs symmetrical (probably non-mosaic), long (♂) antennae, large (♂) colorless (*wh*) ocelli, white eyes and normal male genitalia (Figure 5). Sclerotization of abdomen, however, showed much irregularly distributed female tissue. Tergites were in order from anterior to posterior on left,—♂ ♂ ♂ ♀ ♂ ♂ and on right,—♂ ♀ ♀ ♀ ♀ ♀. Sternites were on left all male and on right,—♀ ♀ ♂ ♀ ♂ ♂ ♂. The gynander reacted as a normal male. Mated with five orange females, it sired 65 orange daughters. From three variegated (*sv wh*) females there were produced females wild type 141, males variegated 57, orange 2 (nos. 934.5, 934.6). This white-eyed gynander



HAPLO-DIPLOID MOSAICS

Figure 8

A—No. 949. A gynander with the paternal eye color mottled, *mo,* in its male ocelli and in regions of the compound eyes. Boundary between these regions and the black biparental female parts is clear-cut showing mottled to be autonomous. Left antenna and abdomen are female, right antenna is male and probably androgenetic. *B*—No. 951. A haplo-diploid male with red-eyed father. A red patch, smokey because non-autonomous, appears in the left eye and the wasp transmitted red in breeding test. The large black ocelli, the black parts of the eyes and the right antenna with fewer segments than the left are diploid male. These parts are male because they are homozygous for sex, receiving *xe* from both parents.

therefore bred as an orange male having gynogenetic *CHB-VI-o* gonads. The two orange sons indicate that *CHB-VI-o* sperm, as well as eggs, have the androgenetic tendency (Figure 6*A*).

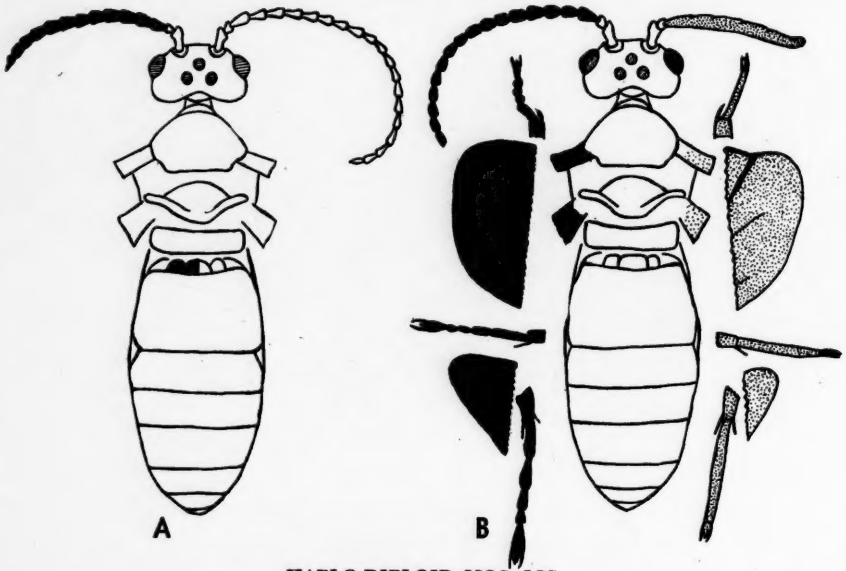
Both egg binuclearity and dispermy are necessary to account for this gynander. There should be two haploid and one diploid nuclei with origin and genes as follows:—♀*o*, ♂*wh*, ♀*o*/♂*wh*.

No. 936, with stock *xc/xd-mo* father, resembled a normal black-eyed male, symmetrical in form and pigmentation and showing superficially no sign of mosaicism. The black eyes marked the specimen as exceptional since diploid males are not to be expected from this cross. When tested with females it failed to react. It acted like a female toward caterpillars attempting to sting them and inspecting them with its long male antennae. Close examination after preservation in alcohol showed the ocellar region to be darker on the left side and the ocelli to be small (♀) and black in agreement with the eyes (♀*o*/♂*wh*). (Figure 6*B*.)

The specimen, a gynander, is of interest because of localization of female region to a portion of the head. Type of reaction is evidently not determined by the antennal sense organs. Whether the body in general is gynogenetic (♀*o*), androgenetic (♂*mo*) or mixed is uncertain.

No. 937, with stock *xc/xd-mo* father, had male head, female abdomen, eyes and ocelli mottled.

No. 938, with stock *xg/xh-wh* father, had orange eyes and large (♂) orange ocelli, left antenna female, right male, abdominal sclerites a confused mixture of male and female, posteriorly male with normal male genitalia. Reactions were male but confused, the specimen flipping vigorously but failing to mate and making occasional attempts to sting the



HAPLO-DIPLOID MOSAICS

Figure 9

A—No. 938. This is to all appearances an "orthodox" gynander with male parts, eyes and ocelli, orange of maternal origin. The left antenna and highly sclerotized regions in the abdomen are biparental female. Had a mating been obtained, it might have revealed paternal white eyes, but this mosaic gives no evidence for androgenesis. *B*—No. 943. A haplo-diploid male mosaic. The right antenna, right legs and right wings are androgenetic showing the paternal trait fused. Tarsi and antenna with fused segments are drawn and also tips of wings. Fused wings are shortened and the primary has an indention near the tip. The left antenna is male but has only sixteen flagellar segments indicating diploidism. Left tarsi and wings are wild type but the distribution of microchaetae on the latter indicates larger cells characteristic of the diploid male. Diploid parts are male because homozygous for sex, receiving *xe* from both parents. Paternal red color is shown dorsally in the left eye and in the large male ocelli. Red is non-autonomous in mosaics, darkening in vicinity of tissue with the dominant allele.

females. This has been called a "wires-crossed" reaction.[9] (Figure 9*A*.)

Nos. 939, 940, with stock *xc/xd-mo* fathers, were at first thought to be mottled males. Eyes and ocelli were typical for mottled, genitalia normal. Examination subsequent to preservation revealed that they were both gynanders with small amounts of female tissue. No. 939 had black color of mesosternum more restricted on left (probably female) and first abdominal tergite definitely female on left. No. 940 had ocellar region lighter on right (♀?), slight asymmetry of fifth abdominal tergite, mesosternum lighter on right (♀?), first three abdominal sternites female on right. The lighter sides in case of asymmetry of nos. 939 and 940 may be female, or the asymmetry may in part indicate gynogenetic male tissue.

Nos. 941, 942, with stock *xc/xd-mo* fathers, might be mistaken for black-eyed biparental males, but the cross should produce none of these. The preserved specimens proved to be symmetrical in color and form. Wing microchaetae indicated haploid males. Although the eyes were black (presumably female), the ocelli, of large (♂) size, were pale but with some dark pigment. Color and size of ocelli suggest that they are gynogenetic male, genetically orange, a non-autonomous character darkened somewhat by influence from the nearby non-orange region.

No. 943, with red fused father from stock *xe/xf-rd* and with diploid brothers, is a haplo-diploid male. Left antenna has 16 flagellar segments (diploid male), right is fused or mixed (7 ± segments), eyes are wild type excent for grading red region dorsally in left; ocelli are light but show some dark pigment, probably modified red (non-autonomous); left legs are wild type, right fused or mixed; both right wings have microchaetae closer together and are smaller (haploid and fused) than left (diploid and wild type); pigmentation is symmetrical; abdomen normal male. (Figure 9*B*.)

No. 949, with stock *xc/xd-mo* father, has female left antenna, male right; large (♂) colorless (*mo*) ocelli; left eye black (♀) with small mottled spot dorsally (♂), right eye mottled (♂) with small black spot dorsally (♀). The clear-cut margin between wild type and mottled indicates mottled to be autonomous. There is no asymmetry in form or color of body. The abdomen is entirely female. Tests showed typical male reactions to females and indifference to caterpillars. (Figure 8*A*.)

No. 950, with stock *xg/xh-wh* father, has left antenna female (13 flagellar segments), right apparently mixed (17 ± showing fusion). Ocelli are large (♂) and colorless (*wh*). Eyes are black (♀) except for white spot in right (Figure 7). The specimen is otherwise entirely symmetrical, abdomen entirely female.

No. 951, with stock *xe/xf-rd* father and diploid brothers, is a haplo-diploid male. Left antenna has 19 flagellar segments (probably haploid), right 17 (probably diploid); ocelli are large (♂) black (diploid), eyes black (diploid) except anterior region of left which is red (haploid). The specimen is otherwise symmetrical with wing microchaetae as in haploid male. It bred as red, 89 sperm carrying red, 126 the dominant allele to orange. (Figure 8*B*.)

Conclusions

As has been stated in previous publications, [9,15] sex mosaics furnish valuable material for identifying the structural basis of reproductive reactions. Thus it has been shown that the head is the determining factor, regardless of the abdomen. Data herewith reported are consistent with this principle. The gynander with visibly female parts restricted to the eyes (no. 936) excludes the antennae as an effective agent since its reactions were definitely female, the antennae male. This would seem to limit the basis of reproductive behavior difference to the brain.

Mosaics are of value in determining autonomy of various traits.[8] Mutant eye colors have thus been shown to develop according to their own genotype, regardless of the presence of the wild type allele in adjoining tissue, except that red, *rd*,[13] and the alleles orange, *o*, and ivory, o^1, are darkened. Among the mosaics herewith reported white is autonomous (nos. 935, 950), while red (nos. 943, 951) and orange (nos. 941, 942) are modified as previously shown. Mottled is now for the first time clearly proved autonomous (no. 949).

Distribution of the two types of nuclei in an egg forming a gynander has usually been such that fertile gynanders have been very infrequent.[15] Two with male instincts and genitalia were unable to mate (nos. 335, 425), two bred as males (nos. 395, 475.5) and two as females (nos. 448, 503). These six had heads of mixed sex and considerable body tissue of sex opposite to their instincts and genitalia. They represent a selection therefore from a considerable number in which proportion of male and female tissue fluctuates about equality.

Six of the eleven gynanders herewith reported had proportion of female tissue very much restricted, with heads and genitalia similarly male (nos. 935, 936, 939, 940, 941, 942). One of the others also (no. 938) was male in genitalia and instincts (confused) but had a mixed head. Only four (nos. 934.3, 937, 949, 950) had a considerable amount of female tissue. This evidence suggests either that the sperm nucleus has an advantage in cleavage over the fusion nucleus or that the male regions are in part gynogenetic. In the latter case there would be two haploid nuclei against one diploid at the start and the latter might be further handicapped by being delayed during syngamy.

The one gynander (no. 935) showing both egg binuclearity and dispermy displayed the paternal trait white, but bred like an orange male of the maternal stock. This specimen recalls the "trinucleate" gynander (no. 640) previously reported[10] which was also predominantly male. Possibly the other irregular types described herewith may also have three types of tissue, those appearing as androgenetic males having very restricted diploid regions.

Including the two orange sons (nos. 934.5, 934.6) of no. 935 by variegated mothers, the one mottled male (no. 934) with *CHB-IX-o* mother and ten irregular types with *CHB-VI-o* mothers (nos. 934.2, 934.4, 935, 937, 939, 940, 943, 949, 950, 951), there are thirteen cases of androgenesis among the eighteen irregular types.

The two orange sons of no. 935 are evidence that *CHB-VI-o* sperm as well

as eggs cause androgenesis. Something about the nucleus, as the element common to egg and sperm, may therefore be responsible. There appears to be a delayed or deficient tendency toward syngamy relative to resumption of mitotic activity in cleavage. In this stock the sperm nucleus evidently resembles the egg nucleus in this respect.

Summary

Sex in *Habrobracon* is determined by a single series of allelic factors. Any haploid or homozygous diploid is male, any heterozygous combination is female.

The multiple effects of the sex-linked gene *fused,* and the differential action of modifying factors on these primary fused effects, are discussed.

Haploid mosaic males and diploid impaternate females developing from unfertilized eggs occur irregularly. From fertilized eggs, the most frequent of the irregular types are gynanders with diploid biparental female parts and haploid male parts developing gynogenetically from a second egg nucleus. Hitherto gynanders with haploid male parts developing androgenetically from a second sperm nucleus have been extremely rare although dispermy may occur in about one per cent of fertilized eggs. An independent sperm nucleus cannot then in general undergo cleavage as readily as an independent egg nucleus.

The present paper reports and describes 18 irregular types produced from crosses of a stock recently obtained. It is suggested that there is in this stock some genetic differences which affect the sperm nucleus making it resemble the egg nucleus in being capable of entering upon cleavage without syngamy.

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TABLE II. Types of offspring expected from normal uninucleate eggs and from binucleate (1%) eggs, unfertilized (1) or fertilized by a single sperm (monospermic) or by two sperm (dispermic) (1%) which may carry a sex allele different from (three alleles involved) (2) or similar to (two alleles involved) (3) the two alleles in the mother. Male parts of offspring may be diploid biparental (*B*) or, if haploid, either gynogenetic (*G*) or androgenetic (*A*).

	Unfertilized (1)	Fertilized			
		Three alleles (2)		Two alleles (3)	
		Monospermic xo 99%	Dispermic xo xo 1%	Monospermic xa 99%	Dispermic xa xa 1%
Uninucleate 99%					
1/2 xb	haploid ♂	♀	♂	♀ 49.005%	♂ .495% A
1/2 xa				diploid ♂	haplo-diploid ♂
	99% G	98.01%	.99% A	49.005% B	.495% B,A
Binucleate 1%					
1/6 xb xb	haploid mosaic ♂	♂	mosaic ♀	♂	mosaic ♀ .0017%
1/6 xa xa				haplo-diploid ♂	mosaic diploid ♂ .0017% B,B
4/6 xa xb				1/2 .495% B,G	♂
	.01% G,G	.99% G	.01%	1/2 .495% G	.0067% B

TABLE I. Sex-linkage of fused, *fu*. Offspring from white-eyed, *wh*, mothers heterozygous for fused crossed with fused fathers with different sex alleles.

Mothers wh fu + / wh + fu	Haploid (male) offspring from unfertilized eggs.	Formulae of fathers or of sperm		
		+ fu	+ wh fu	+ wh fu
Eggs.		Diploid biparental offspring, black-eyed.		
non-crossover, wh fu + 45%	white ♂	wild type ♂	wild type ♀	wild type ♀
wh wh fu 45%	white fused ♂	fused ♀	fused ♂	fused ♀
crossover wh fu fu 5%	white fused ♂	fused ♂	fused ♀	fused ♀
wh wh + 5%	white ♂	wild type ♀	wild type ♂	wild type ♀

HEREDITARY CONGENITAL FLEXED PASTERNS IN JERSEY CATTLE

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INBREEDING has brought to light several hereditary recessive defects in the Jersey herd of the University of California. The anomalies, previously reported, are as follows: (1) an imperfect epithelial condition that is lethal[5]; (2) a lethal type of recessive achondroplasia different from that found in Telemark cattle[2]; (3) a proportionate type of dwarfism[4]; and (4) a type of congenital cataract.[3]

A fifth hereditary defect that has segregated within the herd is a type of crooked legs involving primarily a flexion of the pastern joint. This abnormality, present at birth, persists for a few days or weeks, but gradually disappears. In this it differs from the bowed pastern in Jersey cattle reported by Atkeson *et al*,[1] due to a joint defect of the hind legs and expressed in adult animals. While always manifested in the front legs, flexed pasterns may infrequently affect the hind legs but to a lesser degree. In no case has the defect been found in the hind legs alone. In the more severe cases the flexion is so severe as to cause the hoofs to turn under, thus bringing the knuckle of the pastern joint in contact with the ground (Figure 10*A*). When the condition is less severe, the pastern joint is flexed to a lesser degree, thus causing the animal to stand high on its toes (Figures 10 and 12). The more severely afflicted calves move about by walking on the knuckles of the flexed pastern joints (Figure 10*A*). Although most of the disfunction is associated with the pastern, some have other slight derangements such as stiffness and crookedness in the knee joint. The defect is bilateral, but may not be expressed identically on each side. Likewise, animals may differ considerably in their expression of the character.

The defect is easily recognized. In new-born calves the condition may vary between the two extremes shown in the top photographs of Figure 10. As the afflicted calf grows older, the flexion gradually becomes less severe, until the legs become normal (Figure 10*C*). The time required for recovery apparently depends upon the severity of the condition at birth. The milder cases recovered within the first week of age; the more severe, in six to eight weeks (Figure 10*C*).

Calves manifesting this defect are normal in all other respects. Only the extreme cases require special assistance during the nursing period. Given such assistance, these show no higher mortality than their normal sibs. If, however, the more severely affected calves did not receive special care, the defect would then act as a semi-lethal.

Inheritance

The first record of this anomaly in the University herd was made on April 7, 1931. The calf, 428B, was described as having "deformed legs." Although at least a similar defect had been observed earlier, it had not been recorded. Thereafter, each calf was inspected at birth, and systematic notes were kept on all abnormal cases. A photographic record showing the progressive changes from birth to recovery was made for most of the calves born from 1933 through 1936.

As will be observed from Table I, 320A is the sire and grandsire of the first calf. Since he was used in the herd from 1923 to the latter part of 1930, he probably sired the unrecorded defective calves that were produced before 428B. One can consider him only in a limited way in analyzing the inheritance of flexed pasterns because he produced but three calves after systematic inspection and recording. A complete record,

RECOVERY FROM FLEXED PASTERNS

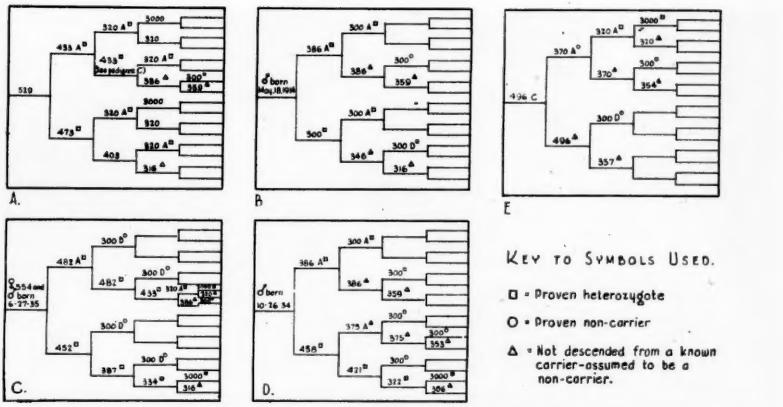
Figure 10

Photographic record of the recovery from flexed pasterns (male calf born June 27, 1935). *A* shows the condition one day after birth. The position of the forelegs is typical. Each hoof is "turned under." *B* shows the improvement five days later. Although there is still considerable flexion, partial recovery permits the calf to stand on its toes. The condition is more severe in the right than in the left forefoot. *C* shows the calf at 65 days of age, all traces of flexion have disappeared.

however, was kept for all progeny of the other sires listed in Table I.

Bull 433A, an inbred son of 320A, sired more calves with flexed pasterns than any other bull. Most of his progeny were produced by mating him to his own daughters and to daughters of 320A (Table I and Figure 11, pedigree *A*). Table II shows the outcome of these matings. Evidently, the observed results can be interpreted on the basis of a single autosomal recessive gene.

Bull 386A produced three calves with flexed pasterns. The pedigree of a male born May 18, 1934, appears in Figure 11, pedigree *B*. Sires 300 and 300*D* in this pedigree are proved non-carriers. Thus not only 386A, but probably also 300A, is heterozygous. Table II shows the outcome of mating 386A to his own daughters and to the daughters of 300A. Here again the results agree with a monofactorial autosomal recessive type of inheritance. Additional evidence supporting but not proving this interpretation is found when heterozygous sire



PEDIGREES OF SIX CALVES EXHIBITING FLEXED PASTERNS

Figure 11

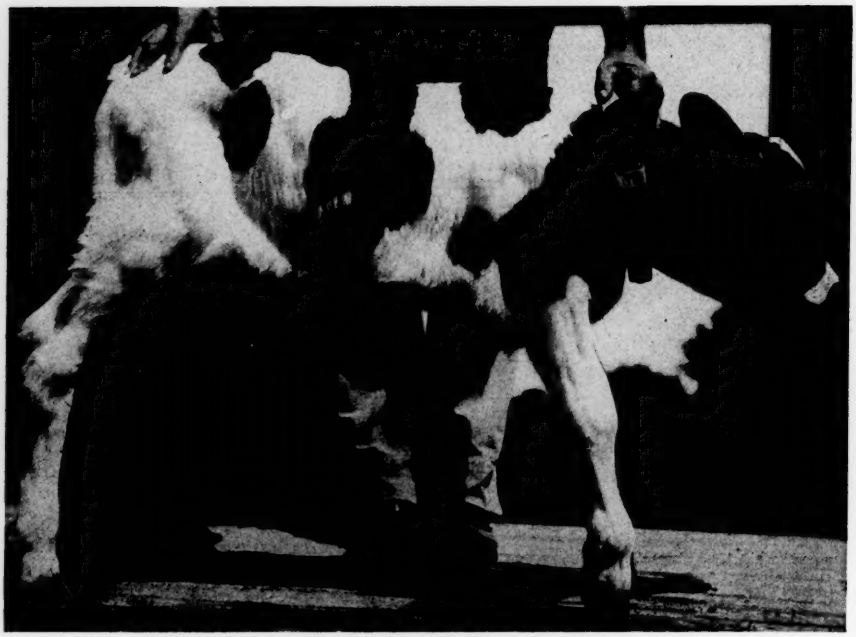
These pedigrees, coupled with progeny tests, illustrate how the absolute or probable genotype of individual animals may be determined. Pedigrees *A* to *D* inclusive are of calves exhibiting flexed pasterns conditioned by a single recessive gene. Pedigree *E* is of a calf that exhibited a probably non-hereditary type of flexed pasterns. (See text.)

433A was twice mated to cow 529, assumed to be homozygous because she exhibited the condition as a calf. Calves 529A and 725 were produced from these matings, and both had flexed pasterns.

The two calves with flexed pasterns sired by 482A out of cow 452 merit discussion (Table I). Both 482A and 452 are inbred progeny of sire 300D, who had been proved by inbreeding to be homozygous normal. Sire 300, also involved in this pedigree, was shown to be homozygous normal. Sire 482A and cow 452 must therefore have obtained the mutant gene from sire 3000, since his son 320A and a daughter 344, out of different cows, are proved heterozygotes (Tables I and II and Figure 11, pedigrees *A* and *C*).

Sire 3000, credited with introducing the gene into the herd, was used from 1917 until 1922. Sire 300A, used ten years later, also carried the gene. These two sires, although not closely related, do have common ancestors in the fifth and sixth generations.

As may be observed from Table I, bull 370A, a son of 320A, produced three male calves with flexed pasterns, 589B being severely and 501B and 496C only mildly affected. The appearance of flexed pasterns in calves 589B and 501B was not surprising, since their sire (370A) and their dams were descended from heterozygous sires. It was surprising, however, that calf 496C had flexed pasterns (Figure 11, pedigree *E*). His dam, 496, was sired by 300D, a proved non-carrier, and she does not trace to a known carrier through her dam. Sire 370A has been used extensively in this herd, having been bred to his own daughters and to daughters of the proved heterozygous sires 320A, 433A, 482A, and 300A. The results of the matings are summarized in Table III. The two defective calves sired by 370A are difficult to explain because they represent so small a proportion of the total offspring resulting from his matings to his own daughters and to daughters of known heterozygous sires. One might assume that 370A is heterozygous and evoke penetrance to explain the deficiency. Since, however, this interpretation is inconsistent with the clear-cut results obtained by mating 433A and 386A to daughters of heterozygous sires (Table II), some other explanation should be sought.



NON-HEREDITARY FLEXED PASTERNS

Figure 12

Holstein calf 162B photographed the second day after birth. The pastern joints are definitely flexed, though not to the extreme illustrated in Figure 10. The calf was completely recovered when seven days old. This non-hereditary type of flexed pastern is phenotypically indistinguishable from the hereditary type.

Perhaps calf 496C (Table I) furnishes a clue. As already mentioned, 496, the dam of 496C, is not descended from any known carrier on either side of her pedigree; in fact her sire is a proved non-carrier. Cow 496 should be homozygous normal. As already noted, two of the three progeny of 370A manifesting the defect were rather mild cases, which recovered more rapidly than did many of the defective progeny sired by either 433A or 386A. Considering all these contingencies, a more logical interpretation is that the cases of flexed pasterns observed in the Jersey herd may be of two kinds. The first type appears to be definitely hereditary and conditioned by one recessive autosomal gene. The second type would then be non-hereditary or developmental in nature. Phenotypically the two cannot be differentiated. On this basis sire 370A would be homozygous normal, and the three defective calves sired by him would represent the non-hereditary type. If this is the correct interpretation, one would expect a low incidence of the non-hereditary type of flexed pasterns in any cattle population. Judging from a study of the progeny of 370A, the incidence of the non-hereditary type of flexed pasterns might be about three per cent or less.

Additional evidence to support the existence of a non-hereditary type of flexed pasterns was found in the University Holstein herd. From April 4, 1931, until his death in 1940, Holstein sire 100B produced 113 offspring when bred to his own daughters. During this period all his calves were carefully observed for

flexed pasterns. Only one, 162B, was observed to show the deformity, in a rather mild form (Figure 12). As the photograph shows, the pasterns are definitely flexed, but not to the degree expressed in the hereditary type for the Jersey calf shown in Figure 10. The Holstein case can be explained satisfactorily on a non-hereditary or developmental basis, the incidence in this case being less than one per cent. Even though the interpretation set forth accounts for all the cases of flexed pasterns that occurred in both the Jersey and the Holstein herds, it may be regarded as a tentative explanation.

Summary

Seventeen calves manifesting a condition of flexed pasterns were born in the Jersey herd of the University of California. The expression of the defect varied. The defect was more often manifested in the front legs alone, though it infrequently affects the hind legs to a lesser degree. In no case was it found in the hind legs alone. In the more severe cases the toes of the front feet were completely turned under. This was never true of the hind feet. In the milder cases the pastern joints were flexed, but the feet were not completely turned under; the animal then walked abnormally on the tips of the toes. Calves exhibiting the condition were as

TABLE II. The observed and expected results of mating heterozygous sires to their own daughters and to daughters of other heterozygous sires, exclusive of cows homozygous for flexed pasterns.

433A mated to his own daughters and to daughters of 320A

	Normal		Defective		
	Male	Female	Male	Female	Total
Observed	21	17	3	3	44
Expected	19.25	19.25	2.75	2.75	
Difference	+ 1.75	− 2.25	+0.25	+0.25	

386A mated to his own daughters and to daughters of 300A

Observed	7	13	1	1	22
Expected	9.6	9.6	1.4	1.4	
Difference	− 2.6	+ 3.4	−0.4	−0.4	

TABLE II. Results of mating 370A to his own daughters and to the daughters of proved heterozygous sires 320A, 433A, 482A, and 300A.

	Normal	Flexed pasterns
Observed	111	2
Expected	98.9	14.1
Difference	+ 12.1	−12.1
$X^2 = 11.7$		

TABLE I. Jersey calves with flexed pasterns.

Herd No.	Sex	Birth date	Sire	Dam	Dam's sire	Remarks
428B	Male	4/7/31	320A	428	320A	Deformed legs.
529	Female	3/9/33	433A	473	320A	Front feet turned under.
536	Female	5/13/33	433A	464	320A	Front feet turned under.
——	Male	5/18/34	386A	500	300A	Stands on toes of left front foot; left hind pastern slightly affected.
——	Male	7/8/34	433A	428	320A	Stands on toes of front feet—slight.
554	Female	7/10/34	482A	452	300D	Stands on toes of front feet; right hind leg slightly crooked.
——	Male	10/26/34	386A	458	375A	Stands on toes of front feet; tendency for feet to turn under.
560	Female	11/7/34	386A	499	300A	Stands on toes of front feet—slight.
585	Female	6/12/35	433A	501	320A	Stands on toes of all four feet.
——	Male	6/27/35	482A	452	300D	Front feet turned under.
529A	Male	9/13/35	433A	529	433A	Stands on toes of front feet—slight.
14U	Male	10/3/35	433A	538	433A	Stands on toes of all four feet; front feet have tendency to turn under.
——	Male	10/28/36	433A	538	433A	Stands on toes of left front foot; right front foot turned under.
725	Female	3/19/37	433A	529	433A	Stands on toes of front feet; have tendency to turn under.
589B	Male	1/10/38	370A	589	370A	Front feet turned under.
501B	Male	10/26/39	370A	501	320A	Stands slightly high on front toes.
496C	Male	9/20/41	370A	496	300D	Stands slightly high on front toes.

vigorous as normal sibs. Since, however, the severely afflicted calves cannot nurse without aid, this defect will act as a semi-lethal unless an attendant is available.

Judging from the results of this study, there may be two types of flexed pasterns. The principal one is conditioned by a single autosomal recessive gene; the other is a non-hereditary type, phenotypically indistinguishable but having an incidence of about two per cent.

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✦

READING BETWEEN THE LINES

FROM time to time a new book appears which seems naturally destined to become the standard text in its field. Such a future may confidently be forecast for the volume under review. This introduction to dermatoglyphics definitely fills a gap, and satisfies the "want of a comprehensive treatise," in any language, on the "patternings of epidermal friction ridges on fingers, palms, toes and soles."

The authors speak with justified assurance, for their own contributions have been large indeed. Over a twenty-year period, they have made their Department the equivalent of the F.B.I. in the field of biological finger print studies. It holds probably the largest collection of prints anywhere assembled for such research; and it is practically an international clearing house for information, and a main center for research and training of personnel, along such lines.

The opening historical and orienting chapters would be enjoyed by anyone. Their popular interest derives mainly from the now widespread realization that finger prints are not rubber stamps or carbon copies, but are a wholly unique and individual possession, poor and small things, to be sure, "but mine own"; something inalienable, to have and to hold, unchanged, from before the cradle to the grave. It is this permanence, and the infinite variety of the pattern details which long ago suggested their use as personal signatures and the simplest reliable means of personal identification.

Since Galton's day practical finger printing has attained a spectacular development, while the scientific side sometimes appeared neglected. The two lines diverged and specialized until at times they have threatened to become mutually unintelligible. Not the least service of the present work is that it includes both aspects, and makes them complement each other. Finger print identification, twin diagnosis, and establishment of paternity, for instance, rest on common basic principles of probability, as developed in Chapters 8, 13 and 14.

"Methodology and Description," in Part II, will be especially welcomed by active workers in this specialty. It is essentially a compact revised manual on the technical aspects of taking prints, tracing the lines of interpretation, describing, formulating and classifying types, and making the standard statistical analyses and comparisons.

The third and largest part, "Biology," will surely hold the interest of Journal readers, for it deals with "the broader and more fundamental significance" of dermatoglyphics to the anatomist, anthropologist, biologist, geneticist, legal expert ,and medical man. There are separate chapters, packed with well organized data, on comparative dermatoglyphics, embryology, symmetry, inheri-

*Cummins, Harold, and Charles Midlo. *Finger Prints, Palms and Soles, an Introduction to Dermatoglyphics.* Pp. 309. $4.00. The Blakiston Company, Philadelphia, 1943.

tance, twin diagnosis, questioned paternity, racial variations, and constitution. Generous acknowledgment is given throughout to pioneers, like the Wilders and Bonnevie, and to the many later investigators.

It may occasion some surprise to note the trends of current researches and to see how much light they throw on problems as diverse as bilateral asymmetry and mirroring, right and left handedness, serial homologies of hands and feet, sexual dimorphism, man's place in the Primate series, racial differences and their geographic distribution, and the time when the first predisposition to certain diseases must have been determined. Being irrevocably fixed during the third and fourth fetal months, dermatoglyphics are good indicators of conditions obtaining in the still earlier critical periods of chemical and histological differentiation. They thus supply a valuable means of estimating the timing, which is important in determining the manner of action of genetic and/or early prenatal environmental factors controlling the development of many abnormalities. Mongolian idiocy, schizophrenia, and neurofibromatosis are among the diseases characterized by more or less distinctive aberrations of dematoglyphic patterns.

Despite their seemingly endless variation from finger to finger, hand to hand, and person to person, the pattern types, ridge counts, and line courses, when once formed are age-stable and environment-stable. They show much resemblance within a family group; and they are much more alike in monozygotic than in dizygotic twins. The general impression gained from the chapter on inheritance is that print features are a complex of a number of inherited, but highly variable, and only partly independent, quantitative characters, which have so far resisted anything more than an approximate mendelian analysis. There are only a few short family pedigrees, mainly showing the presence or absence of some rare pattern, to add to Eugenic Record Office files, or to enrich a Treasury of Human Inheritance.

The book is clearly and interestingly written, remarkably free from errors and ambiguities, abundantly, and for the most part freshly, illustrated, and of superb format. Its broad scope, extensive summaries of relevant research methods, data and theories, and its large and representative bibliography (361 titles) make it an especially helpful general reference work. Everything considered it is several books in one, and all of them are good.

With the present timely publication this newly developed branch of biology may be said to have established itself and come of age.

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WHO CHOOSES WHOM?

Research on the "Social Atom" in Relation to Eugenics

ONE of the basic problems of eugenics, inevitably too long ignored because of a lack of fruitful experimental approaches, is that of choice of marriage partners. Who chooses whom, and why? Until we know more about that, we are left with a large white spot on the eugenic map about which the less said the better. The less said, because what has chiefly come to expression besides vague and general hortations are the too often unconsciously ribald and fundamentally debased (by any decent spiritual yardstick) "rating scales" which are offered as an aid to choosing marriage partners. This is not to deny or belittle the importance of considering the good and the bad in hereditary endowment on the part of any individuals who are contemplating marriage.

One does not edit a genetic journal for a generation without realizing how cruelly genetic defect can torture the lives of sensitive individuals. The poignant letters received asking information (generally "for a friend") regarding the

inheritance of this or that disfiguring or disabling characteristic, often masks utter tragedy when, through the mysterious workings of the choice process a person himself displaying such evidence of pathological heredity (or with relatives who do), chances to fall in love. The stern realities of such a situation brilliantly spotlight the childishness of the score card technique, which pictures the potentially lovelorn taking pencil and pad in hand, and determinedly setting forth to find, by a glorified genetic checkoff, a suitable mate. The one situation is real and has the sombre implications of a Greek tragedy. The other can hardly escape being sophomorish to a degree reached by few other forms of cloistered and theoretical meddling in the lives of human beings.

Hence, it is heartening news for eugenics that we have a progress report based on considerable data of human choice processes.* This report has been years abuilding. In the early thirties this editor saw advertised a book called *Who Shall Survive?,* surely a matter having important eugenic implications, and a review copy was requested. The editor regrets that the book was never reviewed because one could not discover who, if any, were destined to achieve survival. Though fascinated by the black and red arrows which made up something called a "social atom," the editor regretfully decided few readers were likely to be present when the trumpet sounded or the bell tolled, so what the heck? There did not seem to be any genetic implications and yet in a matter of survival, surely there should have been. The inquiry seemed to be an exploration of a basically important eugenic problem, but it seemed not to have jelled to a point where a lay person, sociometrically speaking, could abstract anything but perplexity.

Now, nearly ten years later, we have the next chapter in the progress of sociometry, and this chapter does make sense and establishes a clear linkage with eugenics. Dr. Helen Jennings, who was Dr. Moreno's collaborator in producing *Who Shall Survive?,* has intensively applied the sociometric techniques of choice analysis to a closed human population and has presented the results with elaborate statistical details. The question of who chooses whom begins to be answerable on an experimental basis. The day may not be too far distant when it may be possible to write a eugenic treatise on the choice of mates which is at once dignified, sensible, emotionally satisfying, and genetically sound. When that comes to pass, the discoveries made by the sociometrists may have contributed signally to transforming eugenics from a vague hope to a practical reality.

Choice Processes

The purpose of sociometry is to analyze the pattern of choice and rejection of one personality by another which forms the tenuous network that binds people together into societies. The details of the sociometric technique are given in full in *Leadership and Isolation,* and we will confine ourselves to some of these findings and their eugenic implications. The method has been applied to a number of populations so that we can assume a general validity for the findings. From the eugenic point of view, the story is not complete because the largest group studied in detail is made up of only one sex. The network of choice and rejection between the sexes might be somewhat different. But there seem to be no grounds for assuming that the principles would be greatly changed.

It appears that every individual has a specific choice saturation range. Some will choose for work or play or living together only a small number,—four or five. Others will choose two or three or even five times as many. The pattern of rejection—the "negative choice" of those we definitely would not like to associate with—is also specific for the individual. In a given population we find a small group of isolates

*JENNINGS, HELEN HALL. *Leadership and Isolation.* Pp. XV-240. $3.00. New York: Longmans Green & Co. 1943.

who are either ignored by their fellows or definitely rejected. There is a large middle group of "ordinary citizens"; and floating on top, as it were, a "cream" comprising about 15 per cent of the population made up of the "overchosen." These "overchosen" individuals the author considers are the actual leaders of the group. While they have certain qualities in common they are not a uniform type by many criteria. Extremely varied temperaments are found, and a wide range of I.Q. ratings from the dull-normal to the brilliant. Much the same distribution of I.Q. intelligence is found among the isolates. There is, around the normal range, no significant correlation between intelligence as measured by tests and leadership status. Detailed analysis of this leader group indicates that all members of it contribute significantly in one way or another to the social integration and organization of the group of which they are a part. The author does not analyze the genesis of the personality traits which contribute to inclusion in this group—whether they be inborn or acquired. But it would appear from the context that an important part of the qualities are inherited, for outstanding leader-personalities emerge from very adverse environments. Since all the subjects were drawn from the socially and economically underprivileged population of one of our great States, the development of competent leaders in the social milieu examined is a fact of capital importance.

The analysis of the choice process displayed by members of the group seems to the reviewer to be crucial material for the elaboration of a eugenics program based on the biological and sociological realities of human personality. What *makes* one person choose another and reject a third? The answer to this basic "Why?" of human personality does not emerge in the volume under review. But the analysis of motivations of acceptance and antipathy makes fascinating reading. The existence of deeply rooted traits which condition the reactions of individuals is a basic fact and must be taken into account if eugenics is to be build fruitfully with, rather than across, the grain of human personality.

The question of assortative mating is illuminated by some of the observations. On the whole, the data presented support the view that the choice process tends to build up a stratified population. Among the upper tenth leader group, the proportion of mutual choices was higher than was the choice by leaders from among either the "ordinary citizens" or the "isolates." The leaders rejected no less a proportion of their fellow leaders than they did of the general population, but these rejections represent a higher percentage of mutual reactions. The number of mutual choices among the general population was about four per person. The number of unreciprocated choices ran somewhat higher. It is interesting that the specific rejection of "isolates" by "ordinary citizens" was less great than rejections by the leaders. These intangible walls of rejection, and the saturation of the basic social needs of the individual within the limits of his own stratum, suggest that in a bisex milieu those in the same sociometric categories would tend to choose each other.

As yet we know too little about the extent to which heredity and environment are components in the expression of these choice propensities of adolescents and young adults—the time of life when this matter is of the supremest eugenic importance. The extent to which personality-attributes can be consciously changed is the subject of another recent publication.* Newcomb's study casts important light on the question of how much actual re-orientation in the direction of leadership or of cooperative followership takes place among the members of a college community over the four-year period dur-

*Newcomb, Theodore M. *Personality and Social Change.* Pp. 225. $3.00. Dryden Press, New York. 1943.

ing which freshmen gradually metamorphose into seniors. The major dichotomy studied is progressivism versus conservatism. It is found that there is a shift so large as to be easily measurable from conservative to progressive attitudes among students exposed to the environment of the rather unusual institution studied, Bennington College in Vermont. Some students changed in their attitudes very much more than others. A high correlation was found between I.Q. intelligence and change of attitudes. The non-conservatives tended to be smarter than the conservatives, had better adjustment in their community life and were given much wider recognition as leaders. These findings are interesting to the student of nature versus nurture in demonstrating the possibility, in varying degrees, of changing the attitudes of individuals. If we consider intelligence a specialization to speed adaptive changes of the organism, it is not surprising to find that the more intelligent are the best adapted.

Newcomb's conclusion that the main motivation for change of attitude is a patterning after the prestige group in a college community, points an important moral. The faculty of Bennington College probably averages less conservative than many university faculties. A comparison of one other college community (much more conservative) indicates that less change in attitude took place during the four-year college course. This leads to a suspicion that the more intelligent and progressive elements of the student body are likely to be "sold" eugenics only if the more prestigeful faculty members become affirmatively and challengingly eugenic-minded. The study gives no support to the idea that eugenics can be inculcated merely as a textbook routine. Increased interest and support will come most rapidly from the campus rather than from the classroom.

The approaches to the problem charted in these two volumes open up alluring vistas for further research. We begin to look forward to being able to substitute knowledge for the hopeful exhortations or the pious rationalizations we have had to be content with heretofore. That ordinary human beings appear to exercise their choice processes in a way that by and large advance group welfare is most reassuring. That human beings are fundamentally sound, if the politicians and the reformers and the pressure boys will give them their heads, is tacitly a basic for-granted of both democracy and eugenics. Unless this were true, there would be little point in bothering about either. And these reports do help to strengthen our confidence in the wisdom of the total personality of the run of human beings.—R. C.

BARBARA BURKS MEMORIAL FUND

In the wish to express their appreciation and regard for Barbara Stoddard Burks, whose death has meant a great loss, personal and professional, to many psychologists and geneticists, her friends are establishing a memorial fund. Because of her activities during five years as chairman of the APA Committee on Displaced Foreign Psychologists, it is proposed to use the fund to promote international professional relations among workers in the fields of psychology and genetics—for example, as a loan fund for assistance to European scholars studying or carrying on research in the United States.

For the present the administration of the fund will be in the hands of a committee composed of Gordon Allport, Katherine Brehme, Robert Cook, Kurt Lewin, Theodore Newcomb, Lewis M. Terman, Ruth S. Tolman, and Robert S. Woodworth. Contributions may be sent to Ruth S. Tolman, 4420 Fiftieth St., N. W., Washington 16, D. C.

A FAMILY HISTORY OF DEAFNESS

CYRIL KABAT
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A FAMILY well known to the author has had a history of nineteen cases of deafness occurring in the course of four generations. The time of onset and the symptoms as described by some of the affected individuals themselves and other members of the family are similar to those described for otosclerosis by Davenport, Milles, and Frink.[1] The present age, approximate age at onset, and sex are listed in Table I by the pedigree numbers used in the pedigree (Figure 13). The age at onset is the earliest age at which the affected member or a close relative is positive that deafness was occurring. The solid black pedigree symbols represent an advanced stage in loss of hearing. Incipient stages are represented by the half-black symbols found only in IV-24 and IV-26.

The clinical and audiometric examinations of individuals III-7, III-11, and III-22 by Dr. Meyer S. Fox of Milwaukee are as follows:

> Otological examinations revealed no evidence of any middle ear or ear drum pathology. The audiometer and tuning fork tests showed a bilateral combined type of deafness with negative Rinne tests and slightly increased bone conduction on each side. The history in these cases, clinical findings, audiometer, and tuning fork studies indicate the presence of a bilateral profound deafness of the otosclerotic type.

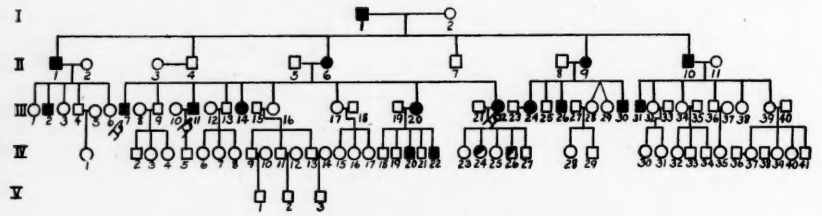
Davenport, *et al.*,[1] explain the inheritance of otosclerotic deafness in the extensive pedigree data which they report in the following manner: "Otosclerosis develops under external conditions that favor it whenever the patient has a constitution that combines two dominant factors as follows: a factor *X* which lies in the sex chromosome and also a factor *A*, which lies in one of the autosomes." This hypothesis leads to the following characteristics in a family history of otosclerosis:

(1) Probability of more affected females than males;
(2) Probability of otosclerosis resulting from non-otosclerotic parents in some cases;
(3) No otosclerotic sons from affected fathers, provided the mother does not carry the sex-linked otosclerotic dominant gene.

In the family reported here there are more affected males than females and still more males than females in the younger age brackets who may later show the condition. There are no cases of otosclerosis resulting from two non-otosclerotic parents, and there are three cases of deaf fathers married to non-deaf mothers producing deaf sons. The frequency with which otosclerotic sons result from deaf fathers and non-deaf mothers is found to be high in the pedigrees given by Davenport, *et al.*,[1] and Chumlea.[2] Therefore it would seem logical to resort to the simpler explanation of a single autosomal dominant gene as the causative agent of deafness in this case. In the pedigree given here and in Chumlea's[2] pedigree no further hypothesis is necessary. In Davenport's[1] pedigrees, however, there are several cases of otosclerotic children resulting from non-otosclerotic parents; the phenomenon of variable penetrance of a dominant

TABLE I. Pedigree Number, Present Age, Approximate Age at Onset, and Sex of Affected Members of the Family

Pedigree Number	Present Age	Age at Onset	Sex
IV-20	19	5	Male
IV-22	21	5	"
IV-26	9	8	"
III-7	35	10	"
IV-24	12	10	Female
III-22	38	13	"
III-14	42	15	"
III-24	42	15	"
III-11	53	15	Male
III-20	49	18	Female
III-26	45	20	Male
III-30	48	20	"
I-1	72	20	"
II-9	81	20	Female
II-6	82	20	"
II-1	72	25	Male
III-2	30	27	"
III-31	40	38	"
II-10	60	58	"



DOMINANT OTOSCLEROSIS

Figure 13

Solid black symbols represent individual showing an advanced stage of hearing loss. Half-black symbols represent incipient deafness in two individuals. Some of the younger individuals in generations IV and V may become deaf later in life.

gene could be used as an explanation of these cases.

On the hypothesis of a single autosomal dominant gene, the expected ratio of normal to deaf resulting from one affected and one normal parent would be 1:1. Actually the ratio of deaf to normal was 19:25. This is a close fit to expectation. When the variation at age of onset is taken into account (Tables I and II), it becomes evident that the ratio is likely to approach expectation even more closely, because some of the progeny classified as normal, particularly those in the lower age brackets (Table II), may become deaf.

It is therefore clear that a single autosomal dominant gene substitution can be used as a satisfactory explanation for the occurrence of deafness in this family.

The author is indebted to Professor A. B. Chapman of the Genetics Department, University of Wisconsin, for his advice and guidance and to Dr. Meyer S. Fox, otologist, Milwaukee, Wisconsin, for the clinical and audiometric examinations.

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TABLE II. Sex, Pedigree Number, Present Age or Age at Death of the Normal Progeny Resulting from the Matings of Deaf and Normal Individuals

Males		Females	
Pedigree Number	Present Age or Age at Death (*)	Pedigree Number	Present Age or Age at Death (*)
IV-27	7	IV-23	5*
IV-18	14	III-38	12*
IV-19	17	IV-25	16
IV-21	20	III-1	25*
IV-5	20	III-32	32
III-9	38	III-3	35
III-36	38	III-34	35
III-4	39	III-39	40
III-25	40	III-6	41
III-13	40	III-17	41
II-7	70*	III-28	42
II-4	75	III-29	42
		III-16	55

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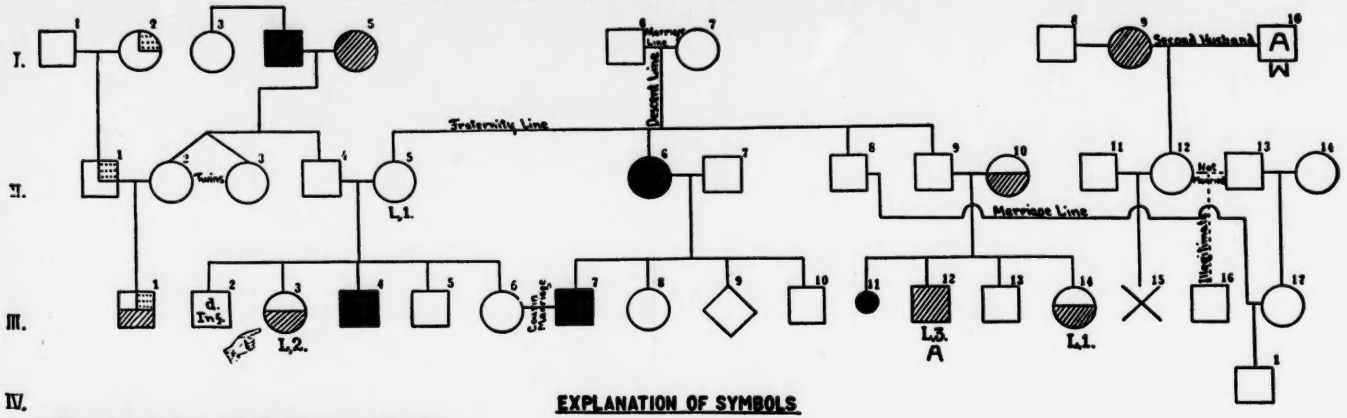

EXPLANATION OF SYMBOLS

(a) Standard for all Pedigree Charts:

□=Male; ○=Female; ◇=Sex unknown; ●=Still-birth or Miscarriage; X=Children — number and sex unknown; ○○=Twins; d. Inf.=Died in Infancy; Points to the Propositus or central figure in the pedigree.

□—○=Parents.

□○□○○=Children.

Roman figures to the left indicate generations; Arabic figures locate individuals, (thus III. 7 is the young man in the third generation who married his cousin).

The following letters, placed in or around the individual's pedigree symbol, are standard for certain traits: A, alcoholic; B, blind; D, deaf; E, epileptic; F, feebleminded; I, insane; M, migraineous; N, normal in reference to traits under consideration; Ne, neurotic; P, paralytic; Sx, sexually immoral; S, syphilitic; T, tuberculous; W, wanderer.

(b) Especially devised or selected for this particular pedigree-chart:

■ ● = Successful leaders in politics. = Extra thumb on right hand. L.1. = Highly successful author. L.3. = Little or no ability in literary efforts. = Superior in vocal music. = Medium attainment in vocal music.

To fit the particular family and traits (whether physical, mental or temperamental; good or bad) under consideration, invent special symbols, or select special letters (in addition to those standardized under (a) above) to be placed within or near the particular individual's pedigree symbol, to indicate particular traits and their degree of development.

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